vitamin C concentrations in the three groups. DHE staining showed higher superoxide production in the coronary vessels of ZO rats than in ZL and Wistar rats. Our results show that ZO might only correspond to early-stage cardiovascular complications associated with MS.

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Cardiac characterization of sgca-null mouse, a model of alpha-sarcoglycanopathy, by using echocardiography

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Alpha-sarcoglycanopathy (LGMD2D) is an autosomal recessive inherited limb-girdle muscular dystrophy caused by mutations in the alpha-sarcoglycan gene, SGCA. Disruption of SGCA gene in mouse (Sgca-null mouse) shares many of clinical pictures observed in patients. Mice disclose progressive muscular dystrophy. We sought to characterize cardiac function in this disease in order to evaluate target therapy. After shaving, Sgca-null mice were anaesthetized with isoflurane before performing echocardiography. Echocardiography was performed with a Vevo 770 Visuals Sonics (30 MHz cardiac probe). Mice were analysed at 5, 13, and 17 months.

Control and Sgca-null mice were similar regarding age and heart rate. At age 5 months, in Sgca-null mice, we found significant anatomical differences regarding the inter-ventricular septal (IVS) wall thickness and the posterior wall (PW) thickness, as compared to their control. The IVS diastolic thickness was significantly increased in Sgca-null mice (0.91mm+/–0.06 vs 0.73 mm+/–0.02; p=0.003). Furthermore, the PW diastolic thickness was also increased in Sgca-null mice compared to WT (0.81mm+/–0.07 vs 0.69 mm+/–0.03; p=0.03). Also, LV mass was significantly increased in Sgca-null mice (147mg+/–15 vs 117.6mg+/–5.3; p=0. 02). At age 13 months, LV mass was significantly higher in Sgca-null mice (LV mass 205.22 mg vs 143 mg; p =0.001).The PW thickness was significantly different in the 2 groups (0.89 mm in Sgca-null vs 0.73 mm; p=0,02). Moreover, Sgca-null mice at 13 months disclosed dilatation of the left ventricle (LVEDD: 4.84mm vs 4.29 mm; p= 0.019). Systolic function was conserved in the 2 groups at 13 months regarding SF and EF.

At age 17 months, we noted a decreasing of the posterior wall thickening (17% vs 30%; p= 0.036) and an increasing of the LV mass/weight (5.6 vs 3.9; p=0,016). No significant differences were found regarding the other anatomic echocardiography parameters.

This study is unique and provides data that will help researchers to evaluate the efficiency of pharmaceutical or gene cardiac therapies.

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Effects of Angiotensin-1 Converting Enzyme inhibition on oxidative stress and bradykinin receptor expression during doxorubic in-induced cardiomyopathy in rats

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To evaluate the mechanisms and the impact of the ACE-inhibitor perindopril (P) in a model of doxorubicin (D)- induced cardiotoxicity, male Wistar rats received D (1mg/kg/d, i.p. for 10 days), P (2 mg/kg/d by gavage from day 1 to day 18), D (for 10 days) + P (for 18 days) or saline.

D decreased systolic blood pressure, body and heart weights. Left ventricular diastolic diameter was increased by D ($p<0.01$) but it was not attenuated by P. D decreased plasma vitamin C ($p<0.05$) and increased the ascorbyl radical/vitamin C ratio ($p<0.01$). This ratio was attenuated by P. No difference was found among groups in cTnI, BNP concentrations and tissue oxidative stress (OS). Myocardial MCP-1 expression was higher in the D group. Cardiac kinin receptor (B1R and B2R) expression was not affected by D yet binding sites for B2R and B1R were increased in D+P and P groups, respectively ($p<0.05$).

In conclusion, D induced cardiac functional alterations, inflammation and plasma OS whereas tissue OS, and cardiac kinin receptors expression were not modified. P did not improve cardiac performance, but modulated kinin receptor expression and enhanced antioxidant defense.

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Lack of association between -308g/a polymorphism of tumor necrosis factor -α gene and myocardial infarction in Tunisian population

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Background: Tumor necrosis factor alpha (TNF-α) is a primary pro-inflammatory cytokine expressed in adipocytes and plays a crucial role in the inflammatory process, it also stimulates the production of chemo-attractant cytokines which may be a key feature of atherosclerosis. Relationship between polymorphisms in TNF-α gene and coronary heart disease has been reported, but remains a controversial subject. The aim of our study was to investigate the possible association between the -308 G/A promoter variant in the TNF-α gene and myocardial infarction (MI) in a sample of Tunisian population.

Design and methods: Our study included 299 male patients MI enrolled from the department of Cardiology at Rabta University Hospital of Tunis and 408 male volunteer subjects with no history of MI. Genomic DNA was extracted from white blood cells, amplified by PCR followed by a digestion with the appropriate restriction endonuclease (***NcoI***) and genotyped by electrophoresis in agarose gels. Statistical analyses were performed using SPSS 11.5.

Results: MI patients compared to controls had significantly higher prevalence of diabetes, hypertension, cigarette smoking and dyslipidemia ($p<0.001$). The genotype frequencies were in agreement with those predicted by the Hardy-Weinberg equilibrium in MI ($X^2=0.137$, $p=0.934$) and control groups ($X^2=0.10$, $p=0.951$). In MI patients the genotype frequencies were 63% for GG, 34.6% for GA and 2.5% for AA, and were 62.2% for GG, 34.8% for GA and 3.0% for AA in controls. No significant difference in genotype and allele frequencies of the TNF-α –308G/A polymorphism were detected between MI and control subjects. In both groups, analyses of variance of lipid levels across –308G/A genotypes showed no significant difference.

Conclusion: Our findings revealed that the -308G/A polymorphism of TNF-α gene is not a relevant marker of MI in the Tunisian population.

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Transient radial myocardial dysfunction in non ischemic segment

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Purpose: In some patients, extensive myocardial dysfunction may be observed during acute coronary syndromes despite limited coronary lesions. Mechanism of depressed contractility during ischemia in remote segments is unclear. In the present study, we addressed the changes in myocardial deformation in remote segments during acute experimental ischemia.